10664603 SM 10/27/08

Amendments to the Specification:

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Please replace the paragraphs beginning at page 23, lines 1-12 with the following amended paragraphs:

Figure 2 shows the PCR primers used for genomic PCR-SSCP of SCN1A (SEQ ID NOs: 99-188);

Figure 3 shows the sequence of the SCN1A mutations found in epilepsy patients (SEQ ID NOs: 189-192 and 309);

Figure 4 shows the PCR primers used for genomic PCR-SSCP of SCN2A (SEQ ID NOs: 193-306);

Figure 5 shows the mutation found in epilepsy patients in SCN2A (SEQ ID NOs: 307 and 308);

Figure 6 shows the PCR primers used for genomic PCR-SSCP of SCN3A (SEQ ID NOs: 310-399); and

Figure 7 shows the mutation found in epilepsy patients in SCN3A (SEQ ID NOs: 400-408).

Please replace the paragraph beginning at page 52, line 3, with the following amended paragraph:

Genomic DNA form IGE and normal patients was obtained by conventional methods. Primers used to amplify the genomic DNA are shown in Figure 2. Following PCR, SSCP analysis was performed and mutations in SCN1A were identified as follows (Figure 3):

(1) Glu1238Asp; normal: GCA TTT GAA GAT ATA; (SEQ ID NO: 189) patient R10191 who has an idiopathic generalized epilepsy (IGE): GCA TTT GAC GAT ATA (SEQ ID NO: 190) found in 1 of 70 IGE patients). The mutation is thus a conservative aa change, in the extracellular domain between III-S1 and III-S2. Furthermore, this residue is located at the junction between the TM domain and the extracellular domain. It may thus influence gating